Complete Summary

GUIDELINE TITLE

HealthPartners Dental Group and Clinics oral cancer guideline.

BIBLIOGRAPHIC SOURCE(S)

HealthPartners Dental Group and Clinics oral cancer guideline. Minneapolis (MN): HealthPartners; 2007 May 2. 20 p. [36 references]

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Oral cancer, including squamous cell carcinoma, melanoma, metastatic neoplasm, Kaposi's sarcoma, and other oral cancers

Note: This guideline deals with the integument of the lips, oral cavity proper and oral pharynx. It does not address salivary glands or connective tissues.

GUIDELINE CATEGORY

Diagnosis
Evaluation
Risk Assessment
Screening

CLINICAL SPECIALTY

Dentistry
Oncology

INTENDED USERS

Dentists

GUIDELINE OBJECTIVE(S)

To provide a model to assess an individual patient's risk for developing an oral cancer and to recommend tools to identify oral cancers earlier in the course of the disease

TARGET POPULATION

Children and adults in the HealthPartners patient population

INTERVENTIONS AND PRACTICES CONSIDERED

1. Risk assessment for oral cancer, including assessment of
    - Tobacco use
    - Alcohol use
    - History of oral cancer
    - Immunodeficiency
    - Sun exposure
    - Age
2. Oral cancer examination including
    - Visual examination or oral soft tissues
    - Visual examination of extraoral head and neck tissues
    - Palpation of head and neck lymph nodes
3. Oral soft tissue biopsy
4. Screening tools such as toluidine blue dye, Vizilite, and brush biopsy
5. Imaging techniques such as computed tomography, magnetic resonance imaging, positron emission tomography, and ultrasonography

MAJOR OUTCOMES CONSIDERED

- Risk for developing oral cancer
- Usefulness of screening tools for oral cancer

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

An online search using Medline, PubMed, current journal articles was conducted.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Not stated

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Not applicable

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Literature was reviewed and discussed by a committee composed of general dentists and one oral surgeon.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

External Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

A draft of the guideline document was sent to expert reviewers for comment.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Risk Factors Associated With the Development of Oral Cancer

There are many factors associated with an increased risk of developing oral cancer. These include tobacco use, alcohol use, past history of oral-pharyngeal cancer, immunodeficiency, sun exposure, and advancing age. There are also synergistic effects among some of these factors.

Tobacco

There is a firm link between tobacco use and the development of oral cancer. The risk increases with the number of cigarettes smoked per day as well as the number of years of tobacco exposure. The risk involves not only cigarettes, but cigars, pipes, and smokeless tobacco products. On average, smokers have 5 to 9 times the risk of developing oral cancer than non-smokers. It appears that the nitrosamines present in tobacco products induce mutations in the genes which suppress cancer. Smokeless tobacco users are 18 times more likely to develop oral lesions (cancerous and non-cancerous) than non-users. 32% of smokeless tobacco users have at least one obvious surface mucosal lesion at the site where the product was actually placed in the oral cavity.

Alcohol

Alcohol intake is associated with an increased risk of oral cancer. Alcohol alone is not carcinogenic for oral squamous cell carcinoma; however it is recognized as a likely promoter of carcinogenesis. As with tobacco, the amount ingested is correlated with risk. The highest risk group comprises those individuals with long term, heavy alcohol use (more than 7 alcoholic drinks per day). Heavy drinkers have up to 15 times the risk of developing oral cancer than abstainers. Moderate alcohol use increases the risk up to 9 times that of non-users. Another effect of heavy alcohol use is an associated decrease in proper nutrition. This may be due to metabolic changes in the liver or lifestyle changes associated with heavy alcohol use, both which result in nutritional deficiencies.

The synergistic effect of alcohol and tobacco use in predisposing one to oral cancer is well known. More than 80% of squamous cell carcinomas of the oral cavity have been associated with the combined use of these products.

Previous History of Oral Cancer

Patients with a previous history of cancer of the oral-pharyngeal mucosa have a 10 to 30% risk of developing a recurrence or a second primary cancer. In light of this, patients with a previous history should be routinely screened for the development of new lesions.

Immune Deficiencies

Impairment of the immune system has shown to increase the risk of developing oral cancer. Along with human immunodeficiency virus (HIV) and other immune disorders, there is some evidence that medically induced immunosuppression can result in oral lesions, for example, following bone marrow or other organ transplants. Advancing age naturally diminishes the immune system's effectiveness which may increase the risk for developing precancerous or malignant oral mucosal lesions.

Sun Exposure

Direct sun exposure is a risk factor for developing squamous cell carcinoma of the lips. As with other risk factors, the effect of the exposure is cumulative. Long term, unprotected exposure to the sun increases the risk of developing actinic cheilitis, a precancerous condition of the vermilion border of the lower lip specifically. Additionally, fair skinned individuals and those living in higher latitudes and elevations have an increased risk for developing lip cancer due to the relatively higher levels of ultraviolet A and B penetration in those environments. However, the clinically accessible nature of the external lip render suspicious lesions associated with sun exposure easier to identify and treat early.

Age

The vast majority of oral cancers are seen in patients over 45 years old. The incidence increases for every decade over 40. As cancer appears to result from repeated or cumulative insults to cells over time, it is not unexpected that increasing age is associated with increased findings of oral cancer. This also correlates with the previously noted decrease in immune system function with advancing age.

Human Papilloma Virus (HPV-16)

HPV-16 has been identified in both pre-cancerous and cancerous lesions of the oral mucosa. It appears that the HPV inactivates a tumor suppressor gene, making one more likely to develop mucosal tumors. These cancers are very often seen in people with no traditional risk factors. Tobacco has little or no relation to these cancers.

Although multiple factors can be identified in the development of oral cancer, it is important to remember that 25% of oral cancers appear in people with no known risk factors.

Oral Cancer Examination

In conducting a visual exam of the oral cavity it is important to be familiar with the spectrum of normal findings so that deviations from normal (like discolorations or lesions) are readily appreciated. Frank squamous cell carcinomas are usually preceded by premalignant surface lesions. Premalignant lesions can be white (leukoplakia), red (erythroplakia), or a combination of white and red (erythroleukoplakia). Although oral mucosal leukoplakia is most often associated with premalignant changes, the red lesions are the ones most likely to

demonstrate precancerous or early malignant changes. The latter lesions and red and white lesions carry the greatest risk of becoming carcinomas.

Oral malignant melanomas usually appear as pigmented lesions of the mucosa. Fortunately, these lesions are extremely rare. It is important to be aware of any newly identified and unexplained pigmented lesion or a pre-existing pigmented lesion that recently changed appearance. Certain benign processes can be mistaken for a melanoma. Among such processes are ethnic pigmentations, amalgam tattoos, and drug-induced pigmentations. The gingival and palate are the highest risk areas for intraoral melanoma.

Kaposi's sarcoma is the most common malignancy associated with acquired immune deficiency syndrome (AIDS). Kaposi's sarcoma is an angioproliferative disease, representing both an inflammatory hyperplasia and a neoplastic process. The skin is the most common site for this cancer but in about half of those with this disease, an oral lesion is observed.

A complete inspection of the oral and oropharyngeal soft tissues and head and neck lymph nodes should be conducted at each dental hygiene exam appointment. An individual determined to be at risk for oral cancer may require a more frequent recall interval than caries or periodontal risks would dictate. It is important to ask the patient if they have noticed any lumps, bumps, bruises or sores that have not healed or experienced any problems with swallowing.

Components of an Oral Cancer Examination

A good oral examination requires an adequate light source, protective gloves, gauze squares, and a mouth mirror.

1. Extraoral examination
    - Inspect head & neck (including the back of the neck)
    - Bimanually palpate lymph nodes and salivary glands
    - Closely inspect the face (including the external ears) for skin lesions
2. Lips
    - Inspect and palpate outer surfaces of lip and vermillion border
    - Inspect and bidigitally palpate inner labial mucosa (upper and lower)
3. Buccal mucosa
    - Inspect and palpate inner cheek lining
4. Alveolar ridge and gingiva
    - Inspect maxillary/mandibular gingiva and alveolar ridges on both the buccal and lingual sides
5. Tongue
    - Have patient protrude tongue and inspect the dorsal surface
    - Have patient lift tongue and inspect ventral surface
    - Grasping tongue with a piece of gauze and gently pulling it out to each side, inspect the lateral borders of the tongue from its tip back to the lingual tonsil region posteriorly
    - Palpate tongue
6. Floor of mouth
    - Inspect and palpate floor of mouth bimanually
7. Hard palate
    - Inspect and palpate hard palate

- Palpate for any lumps

8. Soft palate and oropharynx
    - Gently depress the patient's tongue with a mouth mirror, inspect the soft palate, tonsillar pillars, and oropharynx

Screening and Diagnostic Tools for Oral Cancer

Visual examination of the oral soft tissues, extraoral head and neck tissues and palpation of head and neck lymph nodes is considered the standard of care as part of a complete dental examination. The oral and oropharyngeal tissues lend themselves to visual inspection. One estimate suggests 5 to 10% of routine dental patients have some unusual findings in the oral cavity. Most such findings are benign and reactive in nature, but more rarely a more serious condition like squamous cell carcinoma is detected. A thorough and detailed visual exam along with palpation of tissues is the first step in identifying variations from normal and making an assessment of which conditions pose no threat from those that may lead to more serious consequences.

The biopsy is the gold standard for diagnosing oral cancers. A representative tissue sample obtained surgically and submitted for histopathological examination is the most definitive means of diagnosing oral cancer. A number of screening tools are either commercially available or in development or preparation for distribution. These products purportedly offer help in distinguishing which oral lesions should be biopsied. However, none of them can be relied on to establish definitive diagnoses.

Toluidine blue is a metachromatic dye that has been used for over 40 years to stain tissues suspected of being neoplastic. Toluidine blue stains mitochondrial DNA and cells with greater than normal DNA or altered DNA. It has been found to be useful in selecting sites for biopsy.

ViziLite is a relatively new product developed for evaluating and monitoring oral mucosa abnormalities in populations at increased risk for oral cancer. Acetic acid is applied throughout the mouth and then the tissues are viewed using a special light. In some anatomic regions other than the oral cavity, precancerous lesions appear very opaque-white under this special light. This diagnostic technique has been used successfully in gynecology and is called a colposcopy. However, further study is needed to determine what role, if any, this test should play in screening for oral cancer.

The brush "biopsy", an exfoliative cytologic technique was developed as a means of harvesting a transepithelial sample of cells from an oral surface lesion without having to anesthetize and remove an actual tissue sample (i.e., biopsy specimen) with the scalpel. This, too, is simply a screening tool similar to one that has been used in gynecology for a number of years and is known as a Papanicolaou ("Pap") smear. Many dysplastic lesions are first identified by histpathologically evident changes in the morphology of cells in the epithelial basal cell layer. Therefore, in order to be of use, the brush must obtain cells from this layer. This test can be of used as a preliminary tool in helping to confirm a clinician's suspicion regarding an oral lesion. It must be emphasized that a brush "biopsy" sample analysis does not and cannot provide a definitive diagnosis for oral cancer. A tissue biopsy must be obtained to confirm the diagnosis.

Once a diagnosis of oral cancer has been made, imaging studies may be undertaken to determine the extent of the disease. Current imaging techniques include computed tomography (CT) scan, magnetic resonance imaging (MRI), and positron emission tomography (PET). Ultrasonography may also be useful.

CLINICAL ALGORITHM(S)

An algorithm titled "Flow Chart of Soft Tissue Exam" is provided in the original guideline document.

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of evidence supporting the recommendations is not specifically stated.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Improved assessment and early identification of oral cancers

POTENTIAL HARMS

Not stated

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Audit Criteria/Indicators
Chart Documentation/Checklists/Forms
Clinical Algorithm

For information about availability, see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Staying Healthy

IOM DOMAIN

Effectiveness
Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

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ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2007 May 2

GUIDELINE DEVELOPER(S)

HealthPartners Dental Group - Professional Association

SOURCE(S) OF FUNDING

HealthPartners Dental Group

GUIDELINE COMMITTEE

Not stated

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Not stated

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: None available

Print copies: Available from HealthPartners, 8170 33rd Avenue South, P.O. Box 1309, Minneapolis, MN 55440-1309; Phone: (952) 883-5151; Web site: http://www.healthpartners.com

AVAILABILITY OF COMPANION DOCUMENTS

Potential measures and a sample oral cancer consultation form are provided in the original guideline document.

PATIENT RESOURCES

None available

NGC STATUS

This NGC summary was completed by ECRI Institute on August 8, 2007. The information was verified by the guideline developer on August 28, 2007.

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DISCLAIMER

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